

AMENDMENTS TO THE CLAIMS:

This listing of claims will replace all prior versions and listings of claims in the application:

Claims 1-36 (Cancelled).

Claim 37 (New): A transgenic mouse whose genome comprises a homozygous disruption of the endogenous α -TTP gene, wherein α -TTP expression is inhibited such that the transgenic mouse does not exhibit detectable plasma levels of α -tocopherol.

Claim 38 (New): The transgenic mouse according to claim 37, wherein the mouse is a pregnant female, and wherein the pregnant female fails to maintain pregnancy as assayed by the fetal resorption-gestation test.

Claim 39 (New): The transgenic mouse according to claim 37, wherein the disrupted endogenous α -TTP gene comprises an inserted marker gene.

Claim 40 (New): A method for producing the transgenic mouse according to claim 37, comprising:

(a) inserting a mouse embryonic stem cell into an embryo taken from a pregnant female to form a chimeric embryo, wherein the embryonic stem cell comprises a disrupted endogenous α -TTP gene;

(b) transferring the chimeric embryo into the uterus of a female mouse;

(c) allowing the embryo to undergo full fetal development to term to obtain a mouse comprising the disrupted endogenous α -TTP gene;

(d) crossing a male mouse comprising the disrupted endogenous α -TTP gene with a female mouse comprising the disrupted endogenous α -TTP gene; and

(e) screening the progeny obtained from the cross to identify the mouse according to claim 37.

Claim 41 (New): A transgenic mouse comprising a disrupted endogenous α -TTP gene, wherein α -TTP expression from the disrupted α -TTP allele is inhibited such that transgenic mice homozygous for the disrupted allele exhibit a vitamin E deficiency phenotype.

Claim 42 (New): The transgenic mouse according to claim 41, wherein female transgenic mice homozygous for the disrupted allele fail to maintain pregnancy as assayed by the fetal resorption-gestation test.

Claim 43 (New): The transgenic mouse according to claim 41, wherein the disrupted endogenous α -TTP gene comprises an inserted marker gene.

Claim 44 (New): A method for producing the transgenic mouse according to claim 41, comprising:

(a) inserting a mouse embryonic stem cell into an embryo taken from a pregnant female to form a chimeric embryo, wherein the embryonic stem cell comprises a disrupted endogenous α -TTP gene;

(b) transferring the chimeric embryo into the uterus of a female mouse;

(c) allowing the embryo to undergo full fetal development to term to obtain a mouse comprising the disrupted endogenous α -TTP gene; and

(d) screening the progeny obtained from the cross to identify the mouse according to claim 41.

Claim 45 (New): A method for producing the transgenic mouse according to claim 41, comprising:

(a) inserting a mouse embryonic stem cell into an embryo taken from a pregnant female to form a chimeric embryo, wherein the embryonic stem cell comprises a disrupted endogenous α -TTP gene;

(b) transferring the chimeric embryo into the uterus of a female mouse;

(c) allowing the embryo to undergo full fetal development to term to obtain a mouse comprising the disrupted endogenous α -TTP gene;

(d) crossing a mouse comprising the disrupted endogenous α -TTP gene with a second mouse; and

(e) screening the progeny obtained from the cross to identify the mouse according to claim 41.

Claim 46 (New): A transgenic mouse whose genome comprises a heterozygous disruption of the endogenous α -TTP gene, wherein α -TTP expression from the disrupted α -TTP allele is inhibited such that the transgenic mouse exhibits about one-half the plasma level of α -tocopherol of a corresponding mouse that does not comprise a disrupted endogenous α -TTP gene when the mice are fed with a diet comprising the same amount of α -tocopherol.

Claim 47 (New): The transgenic mouse according to claim 46, wherein the disrupted endogenous α -TTP gene comprises an inserted marker gene.

Claim 48 (New): A method for producing the transgenic mouse according to claim 46, comprising:

- (a) inserting a mouse embryonic stem cell into an embryo taken from a pregnant female to form a chimeric embryo, wherein the embryonic stem cell comprises a disrupted endogenous α -TTP gene;
- (b) transferring the chimeric embryo into the uterus of a female mouse;
- (c) allowing the embryo to undergo full fetal development to term to obtain a mouse comprising the disrupted endogenous α -TTP gene;
- (d) crossing a mouse comprising the disrupted endogenous α -TTP gene with a second mouse; and
- (e) screening the progeny obtained from the cross to identify the mouse according to claim 46.

Claim 49 (New): A method for screening test compounds to identify candidate medicaments for treating diseases characterized by oxidative impairment, comprising:

- (a) providing a transgenic mouse according to claim 37;
- (b) administering the test compound to the mouse; and
- (c) assaying the effect of the test compound on the oxidative impairment of the mouse, to thereby identify test compounds that reduce the oxidative impairment of the mouse as candidate medicaments for treating diseases characterized by oxidative impairment.

Claim 50 (New): A method for screening test compounds to identify candidate medicaments for treating diseases characterized by oxidative impairment, comprising:

- (a) providing a transgenic mouse according to claim 41;
- (b) administering the test compound to the mouse; and
- (c) assaying the effect of the test compound on the oxidative impairment of the mouse, to thereby identify test compounds that reduce the oxidative impairment of the mouse as candidate medicaments for treating diseases characterized by oxidative impairment.

Claim 51 (New): A method for screening test compounds to identify candidate medicaments for treating diseases characterized by oxidative impairment, comprising:

- (a) providing a transgenic mouse according to claim 46;
- (b) administering the test compound to the mouse; and

(c) assaying the effect of the test compound on the oxidative impairment of the mouse, to thereby identify test compounds that reduce the oxidative impairment of the mouse as candidate medicaments for treating diseases characterized by oxidative impairment.